What Happens to Japan if China Catches Cold?

- A causal analysis of the Chinese growth and the Japanese growth

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5.2005

Abstract

Many economic professionals like financial analysts, economic journalists and regulatory officers prevailingly regard the fast growth of the Chinese economy as the key factor that leads recently the Japanese economy to recover from the recession that started since beginning of the nineties. This judgement are mostly underpinned by statistical facts that the Chinese economy grew fast in the last two years; the Japanese export to China has experienced a dramatically increase during last two years, China has become now the biggest foreign trade partner of Japan and so on. However, this convincingly sounding arguments are not sufficient to conclude the statement that the Chinese growth leads Japan out of the recession. In fact the statement has essentially a causal character, which means both the interdependence and the directionality of the dependence. While the positive dependence/correlation between the Chinese economy and the Japanese economy is often explicitly documented by statistical facts, arguments about the directionality of the dependence are totally missing.

In this paper we conduct an empirical study to investigate the directionality of the dependence in order to justify the statement empirically. Taking a probabilistic causal approach, we infer the causal dependence among the Japanese economy and the Chinese economy based on observed data. We find the evidence that the Chinese growth on average has been a positive cause of the Japanese since the later nineties and the temporary positive casual effect is even more pronounced.

JEL CLASSIFICATION SYSTEM FOR JOURNAL ARTICLES:
C1, F4, F4

KEYWORDS: Inferred Causality, Recovery of the Japanese economy.

Contents

1 Introduction

In the keynote address entitled with "China and the Global Economic Recovery" at the American Enterprise Institute Seminar 2005 Anne O. Krueger, First Deputy Managing Director International Monetary Fund, stressed the role of the Chinese growth for the recovery of the world wide recession. This view are shared by many economic professionals, especially with respect to the recovery of the Japanese recession.

Jesper Koll, chief economist for Merrill Lynch in Japan, told the New York Times: "We reckon that 80 percent of the growth in exports in the last 12 months is due to Chinese demand. There is absolutely no question that here in Japan, all eyes are on China."

In A REPORT ON JAPAN, the online journal Globeandmail wrote: "Japan's recovery from long years of slump is coming because so much of what Japanese industry does well complements China's staggering growth. As China's middle classes splash out as consumers, Japan is ready to sell them high-end electronics. As China's industry and constructors demand better and better components, machines and materials, Japan is perfectly placed to turn on the taps."

The believe that the Chinese growth is the most significant factor for the Japanese recovery leads to worry about effect of the slowdown in China. Jesper Koll: "If China slows down, Japan will crash." Morgan Stanley economist Takehiro Sato pointed out in Australian Financial Review that the real danger to Japan was not so much China, but the US economy. "We think that the bulk of export growth to China and Hong Kong since 2001 is ultimately tied to the US market, other than a few exceptions such as steel materials.... If China catches cold, Japan will only catch a sniffle.... [I]f final demand in the US loses steam, the Japanese economy must be ready for another bout of pneumonia."

In this context the journal International Economy conducted an interview of 20 leading economists and high rank managers on the following questions: Is the current economic upswing for once the real thing? If so, to what extent is Japan's expansion too dependent on exports to China? If China experiences a bursting of an economic and financial bubble, to what extent would the Japanese economy be affected? The results were published in Spring, 2004 International Economy.

Either the assertion that the fast growth of the Chinese economy leads the Japanese economy to recover or the concern that the slowdown of the Chinese economy may curb the process of the recovery are all based on one economic hypothesis that the Chinese economy effect the Japanese economy. All the arguments to support this hypothesis are well underpinned by many convincingly sounding statistical facts, they are nevertheless not sufficient.

Because this hypothesis has essentially a causal character, which implies not only the dependence between the Chinese economy and the Japanese economy but also that the directionality of the dependence goes from the Chinese economy to the Japanese economy. The supporting statistics document firstly the interdependence between the Chinese economy and the Japanese economy, and they tell nothing about the directionality. Hence they could also well support the statement that the stop of the Japanese recovery process would lead to the soft landing of the Chinese economy (that is another important issue for many economists [1]).

The concern of this paper is to conduct an empirical causal analysis of the relation between the Chinese economy and the Japanese economy, i.e. we study not only the dependence between them but also the directionality of the dependence between them, in order to provide a solid argument for the hypothesis formulated above. Taking a probabilistic causal approach, we infer the causal dependence among the Japanese economy and the Chinese economy based on observed data. Applying Bayes network technique, we will identify the empirically testable causal order among the variables i.e. identify the directionality of the dependence.

The rest of this paper is organized as follows. Section 2 will give a short introduction to the methodology of inferred causation. We will describe time series causal models that are relevant for dynamic causal analysis. In Section 3 we conduct an empirical investigation of the causal relation between the Chinese economy and the Japanese economy. Section 4 concludes.

[1] See Krueger (2005)

2 The Methodology of Inferred Causation

2.1 Inferred Causation

To the question how statistical methods can be used to conduct causal analysis we consider the following example. Let $X = (x1, x2, x3)'$ be a vector of random variables with jointly normal density function $N(\mu, \Sigma)$. A VAR model in X can be written as follows (the lag length of the VAR is 0).

$$X_t = \mu + \epsilon_t \tag{2.1}$$

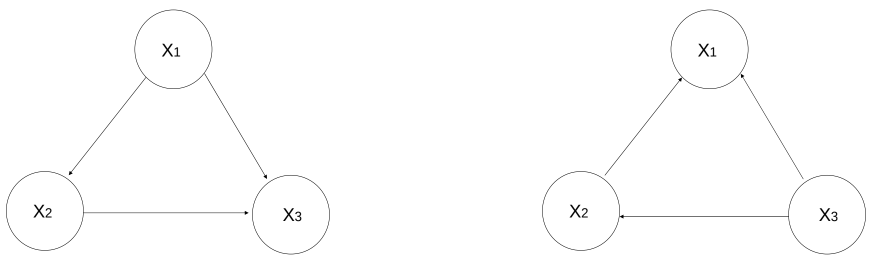
where ϵ_t is the vector of error term with the density function $N(0, \Sigma)$. VAR analysis does not provide any hint on the causal direction among the elements of X_t. Econometrician used to estimate structural VARs which seem to reveal more on the directionality. Applying the Cholesky decomposition of the covariance matrix $\hat{\Sigma} = ADA'$ we get following SVAR model:

$$A^{-1}X_t = \mu_a + \epsilon_t^A, \tag{2.2}$$

where $\epsilon_t^A = A^{-1}\epsilon_t$ are uncorrelated error terms, and A is a low triangular matrix with unit on the principle diagonal. Rewriting this matrix equation (2.2) in its components we have:

$$\begin{aligned} x_{1t} &= \epsilon_{1t}^A \\ x_{2t} &= a_{21}x_{1t} + \epsilon_{2t}^A \\ x_{3t} &= a_{31}x_{1t} + a_{32}x_{2t} + \epsilon_{3t}^A, \end{aligned} \tag{2.3}$$

where a_{ij} is the (i, j) element of inverse of $-A$. System (2.3) seems to suggest us a direction of the influence from $x_{1t} \rightarrow x_{2t} \rightarrow x_{3t}$. One may use a directed graph to present the direction of influence in the SVAR (2.3) as follows (See 1a in Figure 1).



1a 1b

Figure 1: Influence Diagram

However, this inferences of the direction are spurious, because we may equally well get another equivalent (in the sense of identical likelihood) SVAR in which the direction is the other way around: If we apply Cholesky decomposition using the order x_{3t}, x_{2t}, x_{1t} we have another SVAR:

$$\begin{aligned} x_{3t} &= \epsilon^B_{1t} \\ x_{2t} &= b_{21}x_{3t} + \epsilon^B_{2t} \\ x_{1t} &= b_{31}x_{3t} + b_{32}x_{2t} + \epsilon^B_{3t}, \end{aligned} \tag{2.4}$$

where b_{ij} is the i, j element of inverse of $-B$. B is the low triangular matrix of the Cholesky decomposition of Σ in the order of x_{3t}, x_{2t}, x_{1t}.

Now the same observed data seems to suggest another direction of influence: $x_{3t} \to x_{2t} \to x_{1t}$. The corresponding directed diagram is shown in Figure 1b. In fact for an arbitrary order of the components of X_t we may get a corresponding Cholesky decomposition and a corresponding SVAR. Obviously these statistics can not induce any order of influence here. This result should not be surprising, because the only property of the variable X has been that its component are (arbitrarily) jointly normal distributed. It would be a big surprise if we would have got any inference on the direction of influence from statistical analysis of the observations of X for which nothing about the causal structure is assumed.

The SVAR (2.2) and (2.4) can be seen as alternative representations of the jointly distribution as products of conditional distributions:

$$f(x_{1t}, x_{2t}, x_{3t}) = f(x_{3t}|x_{2t}, x_{1t})f(x_{2t}|x_{1t})f(x_{1t}) = f(x_{1t}|x_{2t}, x_{3t})f(x_{2t}|x_{3t})f(x_{3t}).$$

The factorization in conditional distributions can always be presented in a directed graph in the following way: each element of X corresponds to a vertex. A directed edge is drawn from a conditioning element to the conditioned element. Because the factorization corresponds to an order of conditioning among the elements of X, we get a directed acyclic graph(DAG). DAG is also called Bayes network. Both Graph 1a and Graph 1b in Figure 1 are such DAGs.

Because a jointly distribution can always be factorized as products of conditional distributions in any order of its elements, order of factorization alone is not essential for inferring causal directions. We need more information in the data that makes an order outstanding.

Suppose that an additional information is that $f(x_{3t}|x_{1t}) = f(x_{3t})$, which means that the conditional density of x_{3t} given x_{1t} equals to the marginal density of x_{31}. What use can we make of this additional information for causal analysis? Look at the SVAR that corresponds to the Cholesky decomposition in the order of x_{2t}, x_{3t}, x_{1t}, we observe that the corresponding SVAR is as follows:

$$\begin{aligned} x_{1t} &= \epsilon^C_{1t} \\ x_{3t} &= \epsilon^C_{3t} \\ x_{2t} &= c_{21}x_{1t} + c_{23}x_{3t}\epsilon^C_{2t}. \end{aligned} \tag{2.5}$$

The corresponding DAG looks simpler: in Figure 2 there is only two edges instead of three as in Figure 1. Due to the explicit conditional independence the model (2.5) has two parameters, while the model (2.3) and the model (2.4) have three parameters respectively.

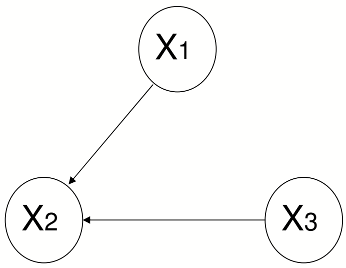


Figure 2: Influence Diagram

According to the principle of parsimony model (2.5) is more preferred. In addition to this parsimony in parameters, model (2.5) suggests that x_{3t} and x_{1t} are causes of x_{2t}. Because these two independent variables become dependent conditional on x_{2t}, x_{2t} must be the effect of both of them.

In the context of the relation between the Chinese economy and the Japanese economy, x_{2t} can be regard as the Japanese growth, x_{3t} the Chinese growth and x_{1t} other factors that may lead Japan out of the recession. If conditioning on these factors x_{1t}, the Japanese growth and the Chinese growth would become independent, it would imply that x_{1t} is the real cause for x_{2t} but not x_{3t}, even if x_{3t} and x_{2t} would have unconditional positive correlation.

The linear recursive structural model (2.5) can be represented in a directed acyclic graph(DAG) as in Figure 2 [2].

The omitted edge between x_{1t} and x_{3t} is due to the conditional (on set $\{\Phi\}$) independence between x_{1t} and x_{3t}. The edges between x_{1t} and x_{2t}, and

[2]For details of the equivalence between linear recursive structural models and DAGs of Bayes network models see Pearl (2000).

between x_{3t} and x_{2t} represent the conditional dependence. The direction of the edges is according to the order of the recursion in the recursive model. A DAG together with the corresponding conditional distribution is called causal model.

It is this causal interpretation of the conditional independence/dependence that makes a model like (2.5) useful for inferencing causality statistically. This approach on inferencing causal order is what we called inferred causation: By analyzing observed data - searching for all possibly conditional independencies - we get one order of variables according to which the conditional independencies among the variables are most explicit. Then we interpret the conditional dependencies as causal dependent and the order as causal order. Because conditional independencies imply zero restrictions on the parameter of the corresponding conditional distribution function, this order implies a most parsimonious representation of the joint distribution. In this context, statistically searching for causal order is equivalent to searching for the most parsimonious model in the class of all possible factorization of the joint distribution.

The main research issue in the inferred causation is to develop effective statistical procedure to uncover the causal structure in the data. Pearl (2000) and Spirtes, Glymour, and Scheines (2001) provide the most detailed up to date results in this area.

2.2 Learning Bayes Networks

As stated in Section 1, inferring causal relation on a set of variables is to select the most acrimonious recursive model within the class of all possible recursive models. Principally, we could analyze every possible recursive model and pick out the most parsimonious one. This is, however, only practicable if the number of variables is very small, because the number of all possible causal models grow explosively with the increase of the number of variables. For a system of 8 variables there are $8! = 40320$ possible models. Even the most powerful computers will run into their computational limit with the increase of variables in the system.

To solve this problem many search algorithms are developed . Hoover (2005) gives a very intuitive description of this procedure. According to Pearl (2000) P.50 the following IC algorithm can be use to find out the true data generating causal model.

IC Algorithm (Inductive Causation)

Input: P a stable distribution on a set X of variables.
Output: a pattern (DAG) compatible with P.

- for each pair of variables X_i and X_j in X, search a set S_{ij} such that $(X_i \perp X_j | S_{ij})$ holds in P. Construct an undirected graph G such that vertices X_i and X_j are connected with an edge if and only if no such set S_{ij} can be found.
- For each pair of nonadjacent variables X_i and X_j with a common neighbor X_k, check if $X_k \in S_{ij}$.
  If it is, then continue. If it is not, then add arrowheads pointing at X_k: $(X_i \rightarrow X_k \leftarrow X_j)$.
- In the partially directed graph that results, orient as many of the undirected edges as possible subject to two conditions:(i) the orientation should not create a new v structure; and (ii) the orientation should not create a directed cycle.

Proposition 2.1 (IC algorithm) *IC-algorithm can consistently identify the inferrable causal structure, i.e. for $T \rightarrow \infty$ the probability of recover the inferrable causal structure of the data generating causal models converges to one.*

Proof : See Pearl (2000) and Spirtes et al. (2001). □

It means that if the data generating linear causal model is statistically distinguishable, IC algorithm will uniquely identify the causal order consistently. If the data generating causal model is not statistically distinguishable, IC algorithm will uniquely identify the causal order among the simultaneous causal blocks consistently.

2.3 Time Series Causal Models

Based on the equivalence of DAG and linear recursive structural model[3], it is essential to represent the statistical model for data in a recursive structure in order to give the data a causal interpretation.

As we know an n-dimensional multivariate time series can be generally represented by a sequence of random n-vector $\{X_t\}$ with a discrete index set $t \in I$ and each X_t has n elements indexed by $i \in I$. A linear causal model for the sequence $\{X_t\}$ will be a recursive model of $\{X_t\}$ in its elements (indexed by t and i). Because we have only one observation for each random variables X_t, a lot restrictions have to be imposed on this recursive model to make statistical inference possible. The task is now to formulate reasonable restrictions on the recursive model such that the resulting class of models are

[3]See Pearl (2000) p.27 and Chen and Hsiao (2005).

general enough to encompass most practical useful time series models and restrictive enough to allow statistical assessment.

Following Chen and Hsiao (2005) time series causal models are defined according to the following three set of restrictions.

Definition 2.2 (FTSCM) *A recursive model of time series is call a finite time series causal model, if it satisfies the following three constrains:*

- *temporal causal constraint,*
- *time invariance of temporal casual structure, and*
- *finite temporal causal influence constraint.*

A time series causal model, for example with $P = 2$ temporal causal influence, has the following recursive structure.

$$\begin{pmatrix} A_2 & A_1 & D & 0 & \dots & & \dots & 0 \\ 0 & A_2 & A_1 & D & 0 & & \dots & 0 \\ \vdots & 0 & A_2 & A_1 & D & 0 & \dots & 0 \\ & & \ddots & \ddots & \ddots & \ddots & \ddots & \vdots \\ 0 & & \dots & 0 & A_2 & A_1 & D & 0 \\ 0 & & & \dots & 0 & A_2 & A_1 & D \end{pmatrix} \begin{pmatrix} X_{-1} \\ X_0 \\ X_1 \\ X_2 \\ \vdots \\ X_{T-1} \\ X_T \end{pmatrix} = \begin{pmatrix} \epsilon_1 \\ \epsilon_2 \\ \vdots \\ \epsilon_{T-1} \\ \epsilon_T \end{pmatrix}. \tag{2.6}$$

The causal relations among the time series variables are expressed by the coefficient matrices $D, A_1, A_2, ..., A_p$. D is itself a low triangular matrix and describes the temporary causal relations among the elements of the vector X_t. A_i describes the causal dependence between the elements of X_t and elements of X_{t-i}. Zero elements in the coefficient matrices A_i implies corresponding causal independence.

2.3.1 FTSCMs and VAR Models

Although FTSCMs are motivated by Bayes network and its equivalence to structural models. There is an intimate relation between FTSCMs and VAR models of time series.

Proposition 2.3 *Under the assumption of homoscedasticity an FTSCM has a VAR representation. A VAR corresponds to an FTSCM representation.*

Proof:

A VAR model is denoted as follows:

$$X_t = \sum_{i=1}^{p} \Pi_i X_{t-i} + U_t. \qquad \text{for } i = p+1, p+2, ..., T, \tag{2.7}$$

and $E(U_t U_t) = \Sigma$. Without loss of generality we take $p = 2$.

The t-th row of the matrix equation (2.6) can be written as follows:

$$DX_t + A_1 X_{t-1} + A_2 X_{t-2} = \epsilon_t, \qquad \text{t=p+1,...,T} \tag{2.8}$$

Premultiply inverse of D to both sides of equation (2.8) we get:

$$X_t = -D^{-1} A_1 X_{t-1} - D^{-1} A_2 X_{t-2} + D^{-1} \epsilon_t, \text{t=p+1,...,T}. \tag{2.9}$$

We have $E(D^{-1}\epsilon_t \epsilon_t' D^{-1'}) = D^{-1}\Omega' D^{-1'}$. Under the assumption of homoscedasticity we have:$\Sigma := D^{-1}\Omega_t' D^{-1'}$. It follows that Equation (2.9) is a VAR(p) model.

On the other hand, for any covariance matrix Σ of any VAR model like (2.7) there exists at least one decomposition, for instance the Cholesky decomposition, such that the following holds:

$$\Omega = A_0^{-1} S (A_0^{-1'}), \tag{2.10}$$

where A_0^{-1} is a low triangular matrix and S is a diagonal matrix[4]. Premultiplying (2.7) by the inverse of A_0, we get:

$$A_0 X_t - \sum_{i=1}^{p} A_0 \Pi_i X_{t-i} = A_0 U_t. \tag{2.11}$$

Since $A_0 U_t$ has diagonal covariance matrix, its components are independent. Obviously, together with the initial condition, (2.11) is formally an FTSCM. □

2.3.2 Learning TSCMs

As in the case of causal models for independent data, the most important issue of statistical treatment of FTSCMs is whether we can recover the causal structure from the observed data, if the data are generated by an FTSCM.

[4]Usually Cholesky decomposition is denoted in the way: $\Omega = A_0 S(A_0'$. For our notation is better to denote the lower triangular matrix of the Cholesky decomposition by its inverse that has the interpretation of recursive causal structure.

For the cases of independent data, Spirtes et al. (2001) give several learning algorithms to recover the true causal structure, if it satisfies the minimality and faithfulness condition. We could have directly applied these algorithms if we would have had repeated observations on same time series. But the typical situation in economics is that we have only one observation for each time point.

Our strategy is a two step procedure: We infer the temporary causal structure first, by taking advantage of the temporal causal constraint. In the second step we infer the temporal causal structure. Concretely we estimate an unconstrained VAR model for the data to get a consistent estimate of the covariance matrix $\hat{\Sigma}$ and a consistent estimate of the coefficient matrices $\hat{\Pi}_i$. Using $\hat{\Sigma}$ as input for IC-Algorithm, we search for the causal structure $\hat{A}_0$. After we know the temporary causal structure, we can identify the temporal causal structure by testing the zero restrictions on $\hat{A}_i$. There are many ways to carry out this significance test[5]. A simple way is to reformulate a recursive model by imposing the simultaneous causal restriction identified by the IC-algorithm and estimate this recursive simultaneous equations model by OLS.

$$A_0^0 X_t + \sum_{i=1}^{p} A_i^* X_{t-1} = \epsilon_t, \tag{2.12}$$

where A_0^0 is the temporary causal structural matrix with the zero restrictions identified by IC algorithm and A_i^* is the unconstrain parameter matrix in (2.12). Then the temporal causal restrictions can be identified by testing the significance of each elements in $\hat{A}_i^*$.

Proposition 2.4 (Two step procedure for FTSCMs)

- *If the temporary causal structure of the data generating FTSCM is observational distinguishable, the two step procedure will identify the true causal structure of the FTSCM consistently.*
- *If an FTSCM is observational distinguishable but the temporary causal structure of the data generating FTSCM is observational indistinguishable, the two step procedure with a consistent model selection criterion will uniquely identify the data generating casual model consistently.*
- *If an FTSCM is observationally indistinguishable, then the two step procedure with a consistent model selection criterion will uniquely identify the causal order of the simultaneous causal blocks.*

[5]See Chen and Hsiao (2005) for details.

Proof: By applying a consistent model selection criterion, we can consistently identify the true lag length of the VAR model. The estimated covariance matrix is consistent. Because the estimate of covariance matrix is consistent and the true structure is observational differentiable, IC algorithm will identify the simultaneous causal structure consistently.

We have $\underset{T\to\infty}{\text{plim}}\hat{A}_0 = A_0$ and $\underset{T\to\infty}{\text{plim}}\hat{\Pi}_i = \Pi_i$. It follows that the estimate of the temporal causal structure is consistent:

$$\underset{T\to\infty}{\text{plim}}\hat{A}_i = -\underset{T\to\infty}{\text{plim}}\hat{A}_0\hat{\Pi}_i = -A_0\Pi_i = A_i.$$

Taking the recursive approach, as the recursive model (2.12) nested the causal model, the test of temporal causal structure becomes a problem of significance test in a classic regression model. As OLS is consistent estimator we have $\underset{T\to\infty}{\text{plim}}\hat{A}_i^* = A_i$. As student t test is consistent, we will consistently identify the temporal structure[6].

In case the FTSCM is observational distinguishable and the temporary causal structure is not, we may have many observational candidates for A_0^0 and the true A_0^0 is under these candidates. However under these candidates we will only find one, namely the true A_0^0, for which model (2.12) is most parsimonious as $T \to \infty$[7]. If there were more than one candidates with most parsimonious restrictions as $T \to \infty$, the data generating FTSCM would have a observational equivalent model. This would contradict to the assumption.

□

3 Empirical Analysis of Sino-Japan Economic Data

To carry out a causal analysis of the relation between the Chinese economy and the Japanese economy we focus on the relation between the growth rate of the Japanese GDP and that of the Chinese GDP. To investigate whether the Chinese growth leads Japan out of the recession is in fact to answer the question whether conditioning on other factors that will also lead Japan out of the recession, the Chinese growth is still a significant cause for the

[6] Taking the bootstrap approach, as bootstrap test is a consistent test, we will consistently identify the zero restrictions on the temporal causal structure.

[7] To rule out the special case that the data generating causal models is not the most parsimonious model The concept of faithfulness is introduced. See Pearl (2000) p. 48 and Spirtes et al. (2001) for details.

growth in Japan. These factors can be classified into three groups: the adjustment mechanism of the economy to the recession, the policy instruments that were/are use to steer the economy out of the recession, and the global economy development.

After bursting of the Japanese bubble at the beginning of nineties the economy ran into a negative spiral of recession. The firms in the recession tried to cut cost such that the unit labor cost decreased, which resulted in rise of unemployment and decrease of household income and consequently led to insufficient demand and deflation. This nullified the original effort of reduction of real unit labor cost and firms were forced to cut labor cost further. The negative dynamic spiral came into being[8].

The economic policy of the government that has been instrumentalized through easy monetary policy [9], fiscal policy and government expenditure - especially government consumption and government investment was conducted to steer the economy out of the recession.

Global economic development used to the most important force that drove the Japanese economy to export out of the recession. Although the globalization has changed somewhat the recovery mechanism, the pull of export - in a specific form - the export to China is the central issue here under investigation.

We measure these three groups of factors by the unit labor cost, the rate of inflation, the central bank rate, the government consumption, and the export volume.

3.1 Description of Data

The data of the relevant variables for this empirical investigation are from OECD database.

Variable	Transformation	Mnemonic	Description of the untransformed series
gj_t	$\Delta_4 log(gdp_t)$	461021YSA	Gross domestic product (Japan)
lcj_t	$\Delta_4 log(unitlabourcost_t)$	464341KSA	Unit labor cost
gp_t	$\Delta_4 log(p)$	461021USA	GDP deflator
$ggexpdj_t$	$\Delta log(gexpd)$	461105RSA	growth of the government consumption
grj_t	$\Delta_4 log(r_t)$	466219D	change of the central bank rate
$gexpj_t$	$\Delta_4 log(expj_t)$	461109RSA	growth of real export
$gxrj$	$\Delta_4 log(xrj_t)$	467003D	change of exchange rate JY/USD
gcn	$\Delta_4 log(gdp_t)$	731561O	growth rate of GDP (China)

Table 1: Raw data used for empirical investigation

[8]See Chen, Chiarella, Flaschel, and Semmler (2005) and Chen and flaschel (2005) for detail description of the adverted real wage effect and the Fisher debt deflation mechanism.

[9]For details see Fukui (2004)

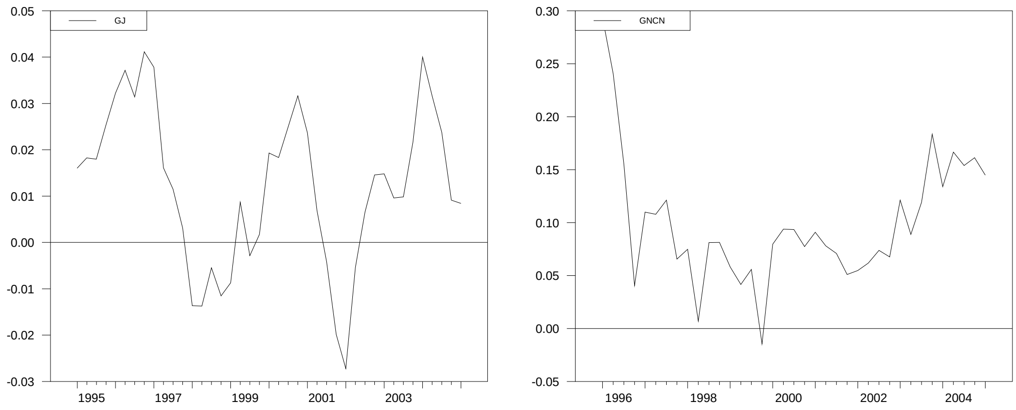


Figure 1: Growth of GDP in Japan and China

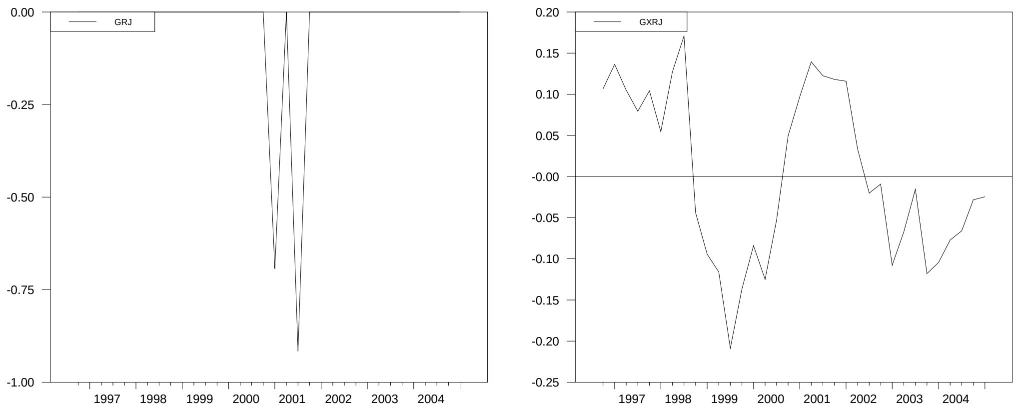


Figure 2: Growth of the Change of central bank rates and the change of exchange rates

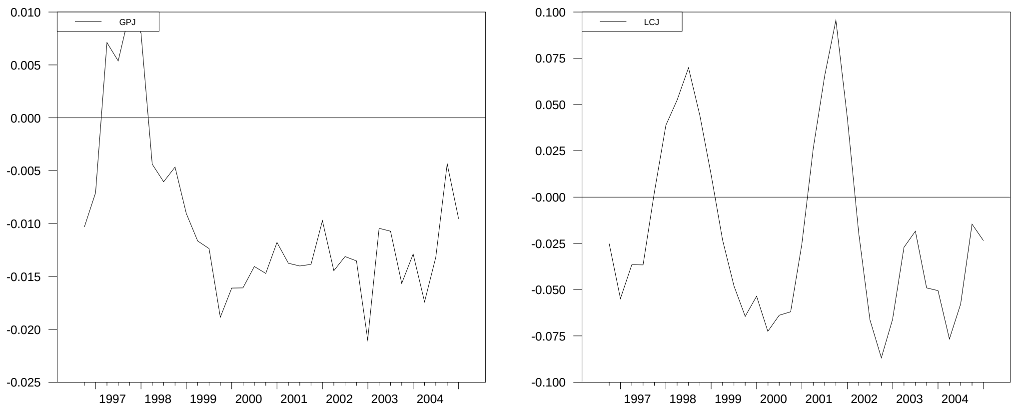


Figure 3: Inflation and change of unit labor cost

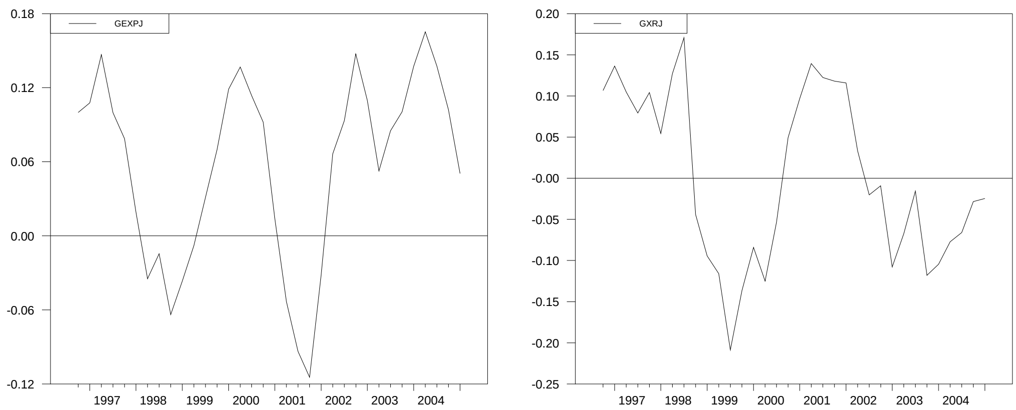


Figure 1: Change of export and change of exchange rate

3.2 Unconditional Approach

Taking the unconditional approach we assume basically the multivariate time series are stationary and ergodic in mean and in covariance. Hence the covariance matrix of the multivariate time series can be estimated by the sample covariance.

Correlation Matrix

gj	gpj	lcj	gxrj	ggexpdj	grj	gncn
1.0000						
-0.2466	1.0000					
-0.7345	0.3449	1.0000				
-0.1953	0.4609	0.5871	1.0000			

0.0624	-0.5087	-0.2056	-0.3897	1.0000		
0.0246	0.1070	-0.2407	-0.2688	-0.0196	1.0000	
0.4747	-0.0134	-0.3384	-0.1993	-0.3170	0.0499	1.0000

Applying IC algorithm provided in Spirtes et al. (2001) we get the following DAG:

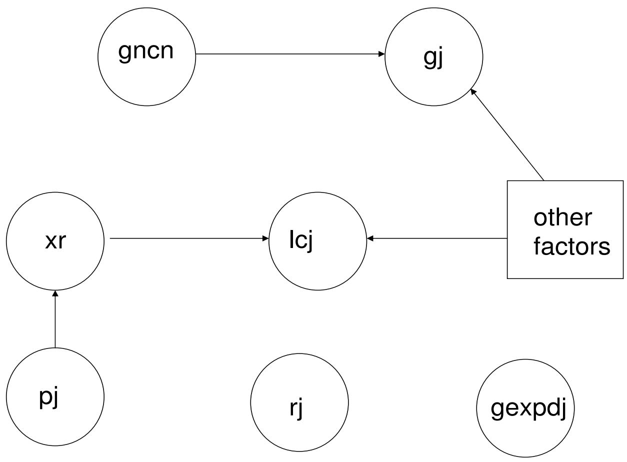


Figure 3: Unconditional Causal Diagram

The causal structure tells us that neither the government consumption nor the central bank rate had significant influence on the growth of the Japanese economy. The deflation and the exchange rate were mutually dependent. There is not evidence about any directionality of dependence between them. However, the exchange rate JY/USD effect positively on the unit labor cost, which implies that devaluation of the Japanese Yen will lead to reduction of unit labor cost. In the economy there were other latent variables that effect on the growth and the unit labor cost.

Beside the other factors that effect on the Japanese growth, there was evidence that in the past 9 years the Chinese economy had a positive impact on the growth of the Japanese economy on average. It can be expected that the further fast growth of the Chinese economy will also have a further positive impact on the recovery of the Japanese economy. However, this evidence is rather a statement of the average effect of the Chinese growth on the Japanese growth. It is also of interesting to know the dynamic aspect of the possible causal structure: whether and how the Chinese growth temporarily and temporally effect the Japanese growth? To this question we apply the FTSCM to infer the dynamic causal structure.

3.3 Dynamic Approach

Taking the dynamic conditional approach we estimated a VAR for *gj*, *pj*, *lcj*, *ggexpdj* and *gncn*. Applying Schwarz information criterion we get the lag length of 4 (see Appendix for details). The correlation matrix of the residuals of the fitted VAR model is as follows.

Correlation Matrix

gj	pj	lcj	ggexpdj	gncn
1.0000				
-0.0745	1.0000			
-0.4496	0.0179	1.0000		
-0.2104	-0.4585	-0.1753	1.0000	
0.5413	-0.1351	0.0681	-0.1267	1.0000

Applying IC algorithm provided in Spirtes et al. (2001) we get the following temporary DAG:

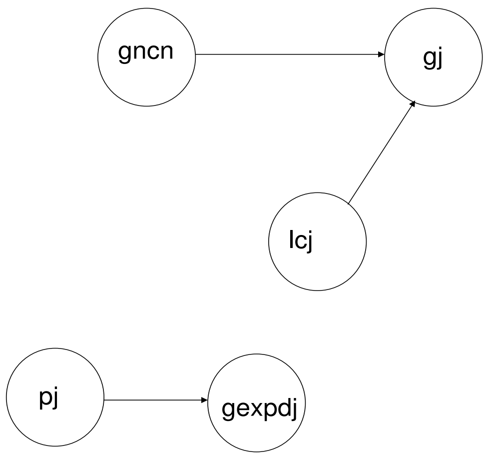


Figure 4: Temporal Causal Diagram

Here the temporary causal influence on the Japanese growth is more pronounced than that the average one. The unit labor cost and the growth of the Chinese economy are identified unambiguously as the two temporary causes for the growth of the Japanese growth. While the decrease of the unit labor cost has positive effect on the Japanese growth, the decrease of the Chinese growth effects negatively on the Japanese growth. Hence the worry about that the slowdown of the Chinese economy may effect the recovery of the Japanese economy is justified.

4 Concluding Remarks

In this paper we investigate the causal relation between the Chinese economy and the Japanese economy. Applying the approach of inferred causation we identify that the Chinese growth effect on that of the Japanese, and this effect is more pronounced in the temporary causal relation than on average. This findings support the hypothesis that the Chinese fast growth will pull Japan out the recession and justify the worry that the slowdown in China may affect the recovery of Japanese economy.

The approach of inferred causation provides a method to identify not only the dependence between two variables but also the directionality of this dependence. This feature is of great interest especially for economists, because the relations between variables formulated by economists are genuine causal. However, for empirical analysis in economics we have to develop causal models for time series, because economical data are in form of time series. Although the two step procedure as described in this paper provides consistent estimation method, simultaneous method is more desirable for both statistical efficiency and consistency in the interpretation of the results.

5 Appendix

```
{
        TETRAD II - Version 1.2 for DOS
                       by
        Peter Spirtes, Richard Scheines,
       Christopher Meek, and Clark Glymour

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Output file: csj3.out
```

Data file: csj3.dat

Parameters:

Sample Size: 34
Continuous Data

Covariance Matrix

gj	gpj	lcj	gxrj	ggexpdj	grj	gncn
1.0000						
-0.2466	1.0000					
-0.7345	0.3449	1.0000				
-0.1953	0.4609	0.5871	1.0000			
0.0624	-0.5087	-0.2056	-0.3897	1.0000		
0.0246	0.1070	-0.2407	-0.2688	-0.0196	1.0000	
0.4747	-0.0134	-0.3384	-0.1993	-0.3170	0.0499	1.0000

Correlation Matrix

gj	gpj	lcj	gxrj	ggexpdj	grj	gncn
1.0000						
-0.2466	1.0000					
-0.7345	0.3449	1.0000				
-0.1953	0.4609	0.5871	1.0000			
0.0624	-0.5087	-0.2056	-0.3897	1.0000		
0.0246	0.1070	-0.2407	-0.2688	-0.0196	1.0000	
0.4747	-0.0134	-0.3384	-0.1993	-0.3170	0.0499	1.0000

P-value for Correlations

gj	gpj	lcj	gxrj	ggexpdj	grj	gncn
0.0000						
0.1615	0.0000					
0.0000	0.0457	0.0000				
0.2704	0.0055	0.0002	0.0000			
0.7281	0.0018	0.2459	0.0216	0.0000		
0.8908	0.5503	0.1726	0.1258	0.9128	0.0000	
0.0043	0.9403	0.0502	0.2609	0.0678	0.7809	0.0000

Significance: 0.0500
Settime: Unbounded

}
{---

```
List of vanishing (partial) correlations that made
TETRAD remove adjacencies.

  Corr. :  Sample (Partial) Correlation
  Prob. :  Probability that the absolute value of the sample
           (partial) correlation exceeds the observed value,
           on the assumption of zero (partial) correlation in
           the population, assuming a multinormal distribution.

Edge             (Partial)
Removed          Correlation                  Corr.    Prob.
-------          -----------                  -----    -----
gj -- gpj        rho(gj gpj)                  -0.2466  0.1615
gj -- gxrj       rho(gj gxrj)                 -0.1953  0.2704
gj -- ggexpdj    rho(gj ggexpdj)               0.0624  0.7281
gj -- grj        rho(gj grj)                   0.0246  0.8908
gpj -- grj       rho(gpj grj)                  0.1070  0.5503
gpj -- gncn      rho(gpj gncn)                -0.0134  0.9403
lcj -- ggexpdj   rho(lcj ggexpdj)             -0.2056  0.2459
lcj -- grj       rho(lcj grj)                 -0.2407  0.1726
lcj -- gncn      rho(lcj gncn)                -0.3384  0.0502
gxrj -- grj      rho(gxrj grj)                -0.2688  0.1258
gxrj -- gncn     rho(gxrj gncn)               -0.1993  0.2609
ggexpdj -- grj   rho(ggexpdj grj)             -0.0196  0.9128
ggexpdj -- gncn  rho(ggexpdj gncn)            -0.3170  0.0678
grj -- gncn      rho(grj gncn)                 0.0499  0.7809
gxrj -- ggexpdj  rho(gxrj ggexpdj . lcj)      -0.3395  0.0530
gpj -- lcj       rho(gpj lcj . gxrj)           0.1034  0.5704
gpj -- gxrj      rho(gpj gxrj . ggexpdj)       0.3313  0.0592

  #: no orientation consistent with assumptions


Significance Level =  0.0500
/Pattern
gj      <> lcj
gncn    -> gj
gpj     -- ggexpdj
gxrj    -> lcj
grj


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        TETRAD II - Version 1.2 for DOS
                      by
        Peter Spirtes, Richard Scheines,
```

```
       Christopher Meek, and Clark Glymour

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Output file: csjd3.out
Data file: csjd3.dat

Parameters:

  Sample Size: 33
  Continuous Data

Covariance Matrix
  gj        gpj       lcj       ggexpdj   gncn
 1.0000
-0.1269    1.0000
-0.4144    0.0112    1.0000
-0.2450   -0.4189   -0.1785    1.0000
 0.4724   -0.1111    0.0635   -0.1071    1.0000


Correlation Matrix
  gj        gpj       lcj       ggexpdj   gncn
 1.0000
-0.1269    1.0000
-0.4144    0.0112    1.0000
-0.2450   -0.4189   -0.1785    1.0000
 0.4724   -0.1111    0.0635   -0.1071    1.0000


P-value for Correlations
  gj        gpj       lcj       ggexpdj   gncn
 0.0000
 0.4848    0.0000
 0.0154    0.9509    0.0000
 0.1717    0.0142    0.3230    0.0000
 0.0049    0.5418    0.7278    0.5566    0.0000


  Significance:      0.0500
  Settime:          Unbounded


}
{-------------------------------------------------------
List of vanishing (partial) correlations that made
```

TETRAD remove adjacencies.

Corr. : Sample (Partial) Correlation
Prob. : Probability that the absolute value of the sample (partial) correlation exceeds the observed value, on the assumption of zero (partial) correlation in the population, assuming a multinormal distribution.

Edge Removed	(Partial) Correlation	Corr.	Prob.
gj -- gpj	rho(gj gpj)	-0.1269	0.4848
gj -- ggexpdj	rho(gj ggexpdj)	-0.2450	0.1717
gpj -- lcj	rho(gpj lcj)	0.0112	0.9509
gpj -- gncn	rho(gpj gncn)	-0.1111	0.5418
lcj -- ggexpdj	rho(lcj ggexpdj)	-0.1785	0.3230
lcj -- gncn	rho(lcj gncn)	0.0635	0.7278
ggexpdj -- gncn	rho(ggexpdj gncn)	-0.1071	0.5566

#: no orientation consistent with assumptions

```
Significance Level =  0.0500
/Pattern
lcj     -> gj
gncn    -> gj
gpj     -- ggexpdj

}
```

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